A STUDY ON ACUTE MYOCARDIAL INFARCTION IN WOMEN

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DECLARATION

I solemnly declare that this dissertation entitled "**A STUDY ON ACUTE MYOCARDIAL INFARCTION IN WOMEN"** at Madras Medical College and Government General Hospital, during 2007-2010 under the guidance and supervision of **Prof.Dr.R.SUKUMAR, M.D.** This dissertation is submitted to the TamilNadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree in General Medicine (Branch-I).

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CERTIFICATE

This is to certify that the dissertation entitled " **A STUDY ON ACUTE MYOCARDIAL INFARCTION IN WOMEN"** is a bonafide work done by **Dr.K.BHARANI**, at Madras Medical College, Chennai in partial fulfillment of the university rules and regulations for award of M.D., Degree in General Medicine (Branch-I) under my guidance and supervision during the academic year 2007 -2010.

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CONTENTS

INTRODUCTION

The twentieth century saw unparalleled increase in life expectancy and a major shift in the cause of illness and death throughout the world. During this transition cardiovascular disease became the most common cause of death worldwide. A century ago CVD accounted for less than 10% of all deaths. Today it accounts for approximately 30% of deaths worldwide including nearly 40% in high-income countries and 28% in low and middle-income countries. Driven by industrialization and associated lifestyle changes, this ongoing transition is occurring around the world among all races, ethnic groups and cultures at an even faster rate than last century[1].

Based on data from Framingham heart study, the lifetime risk of developing symptomatic CAD after age forty is 49% for men and 32% for women. The World Health Organization has estimated that by 2020, the global number of deaths from CAD will have risen from 7.2 million in 2002 to 11.1 million[2]. The evaluation of IHD in women presents a unique and sometimes difficult challenge for clinicians, owing to the difference in symptoms, clinical features and mortality as compared to men. The diagnosis and treatment of CHD have been primarily based on research conducted in men, either excluding women entirely or including limited number of women. The use of traditional risk factors assessment

was limited in prediction of CAD in women[3]. The compendium of coronary heart disease data indicate that current research and strategy development must focus on gender-specific issues in order to address the societal burden and costs related to these demographic shifts in IHD that place women in the majority of those impacted. This significant burden of the disease in women places unique diagnostic, treatment, and financial encumbrances on our society that are only further intensified by a lack of public awareness about the disease on the part of patients and clinicians alike. This societal burden of the disease is, in part, related to our poor understanding of gender-specific pathophysiologic differences in the presentation and prognosis of IHD and the paucity of diagnostic and treatment guidelines tailored to phenotypic differences in women[4].

This study is to analyse the clinical presentation, complications and outcome in those women who presented with myocardial infarction.

AIM OF THE STUDY

1. To analyse the various risk factors in women with acute myocardial infarction.

2. To study the presenting features, site of infarction and complications of acute myocardial infarction in women.

3. To study the outcome in women with acute myocardial infarction admitted to coronary care unit.

REVIEW OF LITERATURE

The clinical presentations of CAD are highly variable. Chest discomfort is usually the predominant symptom in chronic stable angina, unstable angina, micro vascular angina and acute myocardial infarction. However syndromes of CAD also occur in which ischemic chest comfort is absent or not prominent such as asymptomatic myocardial ischemia, congestive heart failure, cardiac arrhythmias and sudden death.

The contemporary approach to patients presenting with ischemic discomfort is to consider that they are experiencing an acute coronary syndrome. The twelve lead ECG is pivotal for segregating patients into those presenting with ST segment elevation and those presenting without ST segment elevation.

Despite the gratifying success of medical therapy for STEMI, several observations indicate considerable room for improvement. Advanced age consistently emerge as one of the principal determinants of mortality in patients with STEMI. Variations have also been observed in the treatment patterns of certain population subgroups with STEMI, notably women[2]. Nevertheless, evidence suggests that the greatest reductions in mortality for elderly patients derive from those strategies employed during the first 24 hours, a time frame in which prompt and appropriate use of life-saving reperfusion therapy has paramount

importance, emphasizing the need to extend advances in drug therapy for STEMI to the elderly.

Revised definition of Myocardial Infarction Criteria for Acute, Evolving or Recent MI[2]

Either of the following criteria satisfies the diagnosis for acute, evolving or recent MI:

1. Typical rise and/ or fall of biochemical markers of myocardial necrosis with atleast one of the following:

    a) Ischemic symptoms

    b) Development of pathological Q waves in ECG

    c) ECG changes indicative of ischemia (ST segment elevation or depression)

    d) Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

2. Pathological findings of an acute myocardial infarction.

Pathology

Almost all MI's result from coronary atherosclerosis, generally with superimposed coronary thrombosis. Non atherogenic forms of coronary artery disease are uncommon.

Causes of myocardial infarction without coronary atherosclerosis:

1. Arteritis
2. Trauma to coronary arteries
3. Coronary mural thickening with metabolic disease or intimal proliferative disease
4. Spasm of coronary arteries
5. Emboli
6. Congenital coronary artery anomalies
7. Myocardial oxygen demand – supply disproportion
8. Hematological (in situ thrombosis)
9. Miscellaneous: cocaine abuse, myocardial contusion

Atherothrombosis:

Atherothrombosis can no longer be considered a disease of the developed world because of myocardial infarction and stroke is increasingly prevalent worldwide across all socioeconomic strata.

Conventional risk factors:

1. Smoking:

Other than advanced age, smoking is the single most important risk factor for coronary artery disease. Ischemic heart disease causes 35 to 40 percent of all smoking related deaths, with an additional 8 percent attributable to second hand smoke exposure. An exceptionally consistent series of studies suggest that, compared with nonsmokers, persons who consume 20 or more cigarettes daily have a two to three fold increase in fatal coronary artery disease. Historically, cigarette consumption was prevalent in men, however this gender gap has markedly narrowed, with overall consumption rates in women is excess of 20 percent.

2. Hypertension:

High blood pressure often confers silent cardiovascular risk and its prevalence is steadily increasing. Of the estimated 50 million people with high blood pressure, almost one third evade diagnosis and only one fourth

receive effective treatment. Hypertension prevalence increased with age (reaching more than 65 percent after the age of 60 years) and tended to be more prevalent in women than men. In the Framingham heart study, even high normal blood pressure (systolic blood pressure 130 to 139 mm Hg, diastolic blood pressure 85 to 89 mm Hg or both) augments risk of cardiovascular disease two fold compared with lower levels.

The most recent report from the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure (JNC VII) has continued appropriately to stress weight control, adoption of the DASH diet with sodium restriction, increased intake of potassium and calcium rich foods, moderate alcohol consumption to fewer than two drinks daily and increased physical activity.

Staging of office blood pressure:

Blood pressure stage	Systolic BP	Diastolic BP
Normal	<120 mm Hg	<80 mm Hg
Prehypertension	120 – 139 mm Hg	80 – 89 mm Hg
Stage 1 hypertension	140 – 159 mm Hg	90 – 99 mm Hg
Stage 2 hypertension	≥160 mm Hg	≥100 mm Hg
Isolated systolic hypertension	>140 mm Hg	<90 mm Hg

3. Hyperlipidemia and elevated low density lipoprotein cholesterol:

The emergence of data from prospective cohort studies such as Framingham heart study, The multiple risk factor intervention trial (MRFIT), Atherosclerosis risk in communities (ARIC) study established the concept of cholesterol as a culprit in coronary heart disease.

4. High density lipoprotein cholesterol, apolipoproteins and other lipid subclasses:

Abundant prospective cohort studies have demonstrated a strong inverse relationship between HDL cholesterol and vascular risk. Despite evidence favouring apolipoprotein AI and B 100 as replacements for HDL and LDL cholesterol, there remains little clinical data that use of these measures improve overall risk prediction.

5. Triglyceride rich lipoprotein:

The role of triglyceride in atherogenesis remains controversial. A cautious approach to triglyceride reduction would seem prudent, because randomized trial data using fenofibrate among diabetic patients with elevated triglyceride have failed to find significant reduction in risk using this approach.

6. Metabolic syndrome, insulin resistance and diabetes:

Insulin resistance and diabetes rank among the major cardiovascular risk factors. Insulin resistance itself promotes atherosclerosis even before it produces frank diabetes.

The definition adopted by National cholesterol education program adult treatment panel regains atleast three of the following five criteria

1. Waist circumference >102 cm in men and >88 cm in women
2. Serum triglycerides of atleast 150 mg/dl
3. HDL cholesterol <40 mg/dl in men and <50 mg/dl in women
4. Blood pressure of atleast 130/85 mm Hg
5. Serum glucose concentration of atleast 110 mg/dl

Novel atherosclerotic risk factors:

1. High sensitivity C- reactive protein
2. Hyper homocystinemia
3. Fibrinogen fibrin D dimer
4. Lipoprotein (a)

Pathophysiology:

Ischemic heart disease is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium. It typically occurs when there is an imbalance between myocardial oxygen supply and demand.

Central to an understanding of the pathophysiology of myocardial ischemia is the concept of myocardial supply and demand. The major determinants of myocardial oxygen demand are heart rate, myocardial contractility and myocardial wall tension. About 70% of the total resistance to flow occurs across three sets of arteries.

1. Large epicardial arteries R1
2. Prearteriolar vessels R2
3. Arteriolar and intramyocardial capillary vessels R3

Effects of ischemia:

Regional disturbances of ventricular contractility cause segmental akinesia or in severe cases bulging (dyskinesia) which can greatly reduce myocardial pump function.

When ischemia is transient, it may be associated with angina pectoris; when it is prolonged, it can lead to myocardial necrosis and scarring with or without the clinical picture of acute myocardial infarction. The severity and duration of the imbalance between myocardial oxygen supply and demand determine whether the damage is reversible or whether it is permenant, with subsequent myocardial necrosis.

Ischemia also causes characteristic changes in the electrocardiogram as evidenced by inversion of T waves, when more severe by displacement of ST segment. Transient T wave inversion likely reflects non transmural, intramyocardial ischemia. Transient ST segment depression often reflects subendocardial ischemia and ST segment elevation is thought to be caused by more severe transmural ischemia.

Another important consequence of myocardial ischemia is electrical instability, which may lead to isolated ventricular premature beats or even ventricular tachycardia or ventricular fibrillation. Most patients who die suddenly from IHD do as a result of ischemia induced tachyarrhythmia.

Clinical features:

Patients with IHD fall into two large group

1. Patients with chronic coronary artery disease who most commonly present with stable angina

2. Patients with acute coronary syndrome composed of patients with acute MI with ST segment elevation on their presenting ECG (STEMI) and those with unstable angina and Non ST segment elevation (UA/NSTEMI)

Almost one half of patients with UA/NSTEMI are women, while more than three fourths of patients with STEMI are men.

Stable angina pectoris

The typical patient with angina is a man older than 50 years or a woman older than 60 years of age who complain of chest discomfort usually described as heaviness, pressure, squeezing, smothering or choking and only rarely as frank pain. When the patient is asked to localize the sensation, he or she will typically place their hand over the sternum, sometimes with a clenched fist (Levine's sign).

Angina is usually crescendo decrescendo in nature typically lasts 2-5 minutes and radiate to either shoulder and to both arms.

However, especially in women and diabetics, angina pectoris may be atypical in location. The typical textbook symptoms of substernal chest pain or pressure radiating to the arms may not be the primary symptoms in women presenting with CAD. Angina equivalents are symptoms of myocardial ischemia other than angina. These include

dyspnea, nausea, fatigue and faintness and are more common in the elderly, women and in diabetic patients.The new onset of symptoms and their relationship to activity has more clinical relevance than their physical location. Women report symptoms more often during daily activities and mental stress than during exercise[5].

Unstable angina:

Unstable angina is defined as angina pectoris or equivalent ischemic discomfort with atleast one of the three features[1]

1. It occurs at rest (or with minimal exertion) usually lasting >10 minutes

2. It is severe and of new onset (i.e. Within the prior 4-6 weeks)

3. It occurs with a crescendo pattern (i.e. clinically more severe, prolonged or frequent than previously)

The diagnosis of NSTEMI is established if a patient with clinical features of unstable angina develops evidence of myocardial necrosis, as reflected in elevated cardiac biomarkers.

Clinical presentation of STEMI:

Pain is the most common presenting complaint in patients with STEMI. It is similar in character to angina pectoris, but commonly occurs at rest, is usually more severe and lasts longer. The pain of STEMI may radiate as high as the occipital area but not below the umbilicus. However STEMI presents without pain in elderly, women and diabetics in whom it can present as pulmonary edema, loss of consciousness, appearance of arrhythmia or merely an unexplained drop in arterial pressure[3].

Physical findings:

Although many patients have a normal pulse rate and blood pressure within the first hour of STEMI, about one fourth of patients with anterior infarction have manifestations of sympathetic nervous system hyperactivity such as tachycardia and/or hypertension. Upto one half of patients with inferior infarction show evidence of parasympathetic hyperactivity such as bradycardia and/or hypotension.

1. The precardium is usually quiet and the apical impulse may be difficult to palpate. In patients with anterior wall infarction, an abnormal systolic pulsation caused by dyskinetic bulging of infracted myocardium may develop in

periapical area within the first days of illness and then may resolve.

2. $S_{3,}$ S_4 may be heard.

3. Paradoxical splitting of S_2

4. Transient midsystolic apical systolic murmur due to dysfuntion of the mitral valve apparatus.

5. Pericardial friction rub and decrease in volume of pulse

Laboratory findings:

The laboratory tests of value in confirming the diagnosis may be divided into four groups

1. ECG

2. Serum cardiac biomarkers

3. Cardiac imaging

4. Nonspecific indices of tissue necrosis and inflammation

ECG:

The ECG is a cornerstone in the diagnosis of acute and chronic ischemic heart disease. The findings depend on several key factors: the nature of the process [reversible (i.e., ischemia) versus irreversible (i.e., infarction)], the duration (acute versus chronic), extent (transmural versus subendocardial), and localization (anterior versus inferoposterior), as well as the presence of other underlying abnormalities (ventricular hypertrophy, conduction defects). When the acute ischemia is transmural, the ST vector is usually shifted in the direction of the outer (epicardial) layers, producing ST elevations and sometimes, in the earliest stages of ischemia, tall, positive so-called hyperacute T waves over the ischemic zone. With ischemia confined primarily to the subendocardium, the ST vector typically shifts toward the subendocardium and ventricular cavity, so that overlying (e.g., anterior precordial) leads show ST-segment depression (with ST elevation in lead aVR). Profound ST elevation or depression in multiple leads usually indicates very severe ischemia. From a clinical viewpoint, the division of acute myocardial infarction into ST segment elevation and non-ST elevation types is useful since the efficacy of acute reperfusion therapy is limited to the former group. The ECG leads are more helpful in localizing regions of ST elevation than non-ST elevation ischemia. For example, acute transmural anterior (including apical and lateral) wall ischemia is reflected by ST elevations or increased T-wave positivity in one or more of the precordial leads (V1 to

V6) and leads I and aVL. Inferior wall ischemia produces changes in leads II, III, and aVF. Posterior wall ischemia may be indirectly recognized by reciprocal ST depressions in leads V1 to V3.Prominent reciprocal ST depressions in these leads also occur with certain inferior wall infarcts, particularly those with posterior or lateral wall extension. Right ventricular ischemia usually produces ST elevations in right-sided chest leads. When ischemic ST elevations occur as the earliest sign of acute infarction, they are typically followed within a period ranging from hours to days by evolving T-wave inversions and often by Q waves occurring in the same lead distribution. With infarction, depolarization (QRS) changes often accompany repolarization (ST-T) abnormalities. Necrosis of sufficient myocardial tissue may lead to decreased R-wave amplitude or abnormal Q waves in the anterior or inferior leads. Loss of depolarization forces due to posterior or lateral infarction may cause reciprocal increases in R-wave amplitude in leads V1 and V2 without diagnostic Q waves in any of the conventional leads. Atrial infarction may be associated with PR-segment deviations due to an atrial current of injury, changes in P-wave morphology, or atrial arrhythmias. In the weeks and months following infarction, these ECG changes may persist or begin to resolve. Complete normalization of the ECG following Q-wave infarction is uncommon but may occur, particularly with smaller infarcts. In contrast, ST-segment elevations that persist for several weeks or more after a Q-wave infarct usually correlate with a severe underlying wall motion disorder (akinetic or dyskinetic zone), although not

necessarily a frank ventricular aneurysm. The ECG has important limitations in both sensitivity and specificity in the diagnosis of ischemic heart disease. Although a single normal ECG does not exclude ischemia or even acute infarction, a normal ECG throughout the course of an acute infarct is distinctly uncommon.

Serum cardiac biomarkers:

Certain proteins called serum cardiac biomarkers are released from necrotic heart muscle after STEMI. The temporal pattern of protein release is of diagnostic importance, but contemporary urgent reperfusion strategies necessitate making a decision based largely on a combination of clinical and ECG findings.

Biomarkers such as cardiac specific Troponin T (CTnT) and cardiac specific Troponin I (CTnI) may increase after STEMI to levels >20 times higher than the upper reference limit. They are particularly valuable in differentiating skeletal muscle injury from small MI that may be below the detection limit for CK and CKMB measurements and they are therefore of particular value in distinguishing UA from NSTEMI. Levels of CTnT and CtnI rise as early as 3 hours after STEMI and elevated for 7 to 10 days after STEMI. Creatine phosphokinase rises within 4 to 8 hours and generally return to normal by 48 to 72 hours. A

ratio of CKMB: CK activity ≥2.5 suggests but is not diagnostic of a myocardial rather than a skeletal muscle source for the CKMB elevation.

Cardiac imaging:

Abnormalities of wall motion on two-dimensional echocardiography are almost universally present. Although acute STEMI cannot be distinguished from an old myocardial scar or from acute severe ischemia by echocardiography, the ease and safety of the procedure make its use appealing as a screening tool. In the emergency department setting, early detection of the presence or absence of wall motion abnormalities by echocardiography can aid in management decisions, such as whether the patient should receive reperfusion therapy. Echocardiographic estimation of left ventricular (LV) function is useful prognostically; detection of reduced function serves as an indication for therapy with an angiotensin-converting enzyme inhibitors. Echocardiography may also identify the presence of right ventricular (RV) infarction, ventricular aneurysm, pericardial effusion, and LV thrombus. In addition, Doppler echocardiography is useful in the detection and quantitation of a ventricular septal defect and mitral regurgitation, two serious complications of STEMI. Several radionuclide imaging techniques are available for evaluating patients with suspected STEMI. However, these imaging modalities are used less often than echocardiography because they are more cumbersome and lack sensitivity

and specificity in many clinical circumstances. Myocardial perfusion imaging with 201Tl or99mTc-sestamibi, which are distributed in proportion to myocardial blood flow and concentrated by viable myocardium, reveal a defect (“cold spot”) in most patients during the first few hours after development of a transmural infarct. However, although perfusion scanning is extremely sensitive, it cannot distinguish acute infarcts from chronic scars and thus is not specific for the diagnosis of acute MI. Radionuclide ventriculography, carried out with 99mTc-labeled red blood cells, frequently demonstrates wall motion disorders and reduction in the ventricular ejection fraction in patients with STEMI. While of value in assessing the hemodynamic consequences of infarction and in aiding in the diagnosis of RV infarction when the RV ejection fraction is depressed, this technique is nonspecific, as many cardiac abnormalities other than MI alter the radionuclide ventriculogram.

Management in the emergency department:

The goals for management of patients with suspected STEMI include control of cardiac discomfort, rapid identification of patients who are candidates for urgent reperfusion therapy, triage of low risk patients to their appropriate location in the hospital and avoidance of inappropriate discharge of patients with STEMI.

Aspirin is essential in the management of patients with STEMI. Rapid inhibition of cyclooxygenase in platelets followed by a reduction of thromboxaneA_2 level is achieved by buccal absorption of a chewed 160-325mg tablet. This measure should be followed by daily oral administration of aspirin in a dose of 75- 162mg.

Control of discomfort:

Sublingual nitroglycerin upto three doses of 0.4mg should be administered at about 5 minutes intervals. Other drugs that can be used are morphine, intravenous beta blocker.

A commonly employed regimen is metaprolol 5mg every 2-5 minutes for a total of 3 doses provided the patient has a heart rate >60 beats per minute, systolic pressure >100mmHg, a PR interval >0.24sec and rales that are no high than 10cm up from the diaphragm. Fifteen minutes after the last intravenous dose, an oral regimen is initiated 50mg every 6hrs for 48 hrs, followed by 100mg every 12hrs.

Management strategies:

The primary tool for screening patients and making triage decision is the initial 12 lead ECG. When ST segment elevation of atleast 2mm in two contiguous precordial leads and 1mm in two adjacent limb leads is

present, a patient should be considered a candidate for reperfusion therapy. In the absence of ST segmental elevation, fibrinolysis is not helpful and evidence exists suggesting that it may be harmful.

According to ACC/AHA guidelines for the management of patients with STEMI, transport time to the hospital is variable from case to case, but the goal is to keep total ischemic time within 120 minutes.

There are three possibilities

1. If EMS has fibrinolytic capacity and the patient qualifies for therapy, prehospital fibrinolysis should be started within 30 minutes of EMS arrival.

2. If EMS is not capable of administering prehospital fibrinolysis and the patient is transported to a non PCI capable hospital, the hospital door to needle time should be within 30 minutes.

3. If the patient is transported to a PCI capable hospital, the hospital door to balloon time should be within 90 miutes.

Inter hospital transfer:

It is also appropriate to consider emergency interhospital transfer to a PCI capable hospital for mechanical revascularisation if

1. There is contraindication for fibrinolysis

2. PCI can be initiated promptly within 90 minutes

3. Fibrinolysis is administered and is unsuccessful (i.e. rescue PCI)

Primary PCI:

It appears to be more effective than fibrinolysis. It has the advantage of being applicable to patients who have contraindication to fibrinolytic therapy. Primary PCI is generally preferred when the diagnosis is in doubt, cardiogenic shock is present, bleeding risk is increased or symptoms have been present for atleast 2- 3 hours when the clot is more mature and less easily lysed by fibrinolytic drugs.

Fibrinolysis:

If no contraindications are present fibrinolytic therapy should ideally be initiated within 30 minutes of presentation (i.e. door to needle time ≤30 minutes). The principal goal of fibrinolysis is prompt restoration of full coronary patency. The fibrinolytic agents approved for intravenous use are

1. 1.tPA

2. Streptokinase

3. Tenecteplase

4. Reteplase

Although reduction of mortality rate is more modest, the therapy remains of benefit for many patients seen 3-6 hours after the onset of infarction and some benefit appears to be possible upto 12 hours, especially if chest discomfort is still present and ST segments remain elevated.

tPA – 15mg bolus followed by 50mg intravenously over first 30 minutes followed by 35mg over the next 60 minutes.

Streptokinase – 1.5 million units intravenously over 1 hour.

rPA – double bolus regimen 10MU bolus given over 2- 3 minutes followed by a second 10MU bolus 30 minutes later.

TNK – single iv bolus 0.53mg/kg over 10 seconds.

Contraindications and complications:

Absolute contraindications:

1. Cerebrovascular hemorrhage at any time

2. Non hemorrhagic stroke or other cerebrovascular event within the past year

3. Marked hypertension >180/110mm Hg at anytime during the acute presentation

4. Suspicion of aortic dissection

5. Active internal bleeding (excluding menses)

Relative contraindications:

1. Current use of anticoagulants (INR ≥2)

2. Recent i.e. < 2 weeks of invasive or surgical procedure or prolonged (> 10 minutes) CPR

3. Known bleeding diathesis

4. Pregnancy

5. Hemorrhagic ophthalmic condition

6. Active peptic ulcer disease

7. A history of severe hypertension that is currently adequately controlled.

Because of the risk of an allergic reaction, patients should not receive streptokinase if that agent had been received within the preceding 5 days to 2 years. Hemorrhage is the most frequent and potentially the most serious complication. Hemorrhagic stroke is the most serious complication and occurs in 0.5- 0.9% of patients being treated with these agents. Large scale trials have suggested that the rate of intracranial hemorrhage with tPA or rPA is slightly higher than with streptokinase.

Cardiac catheterisation and coronary angiography should be carried out after fibrinolytic therapy if there is evidence of either

1. Failure of reperfusion (persistent chest pain and ST segment elevation >90 minutes), in which case a rescue PCI should be considered.

2. Coronary artery reocclusion (re-elevation of ST segment and/or recurrent chest pain) or the development of recurrent ischemia (such as recurrent angina in the early hospital course or a positive exercise stress test before discharge) in which case an urgent PCI should be considered.

The potential benefits of routine angiography and elective PCI even in asymptomatic patients following administration of fibrinolytic therapy are controversial, but such an approach may have merit given the

numerous technological advances that have occurred in the catheterisation laboratory. Coronary artery bypass surgery should be reserved for patients whose coronary anatomy is unsuited to PCI, but in whom revascularisation appears to be advisable because of extensive jeopardized myocardium or recurrent ischemia.

Hospital phase management:

Coronary care units:

The duration of stay in the coronary care unit is dictated by the ongoing need for intensive care. If symptoms are controlled with oral therapy, patients may be transferred out of the coronary care units. Also, patients who have confirmed to be at low risk (no prior infarction and no persistent chest discomfort, CHF, hypotension or cardiac arrhythmias) may be safely transferred out of the coronary care unit within 24 hours.

Activity:

Patients with STEMI should be kept at rest for the first 12 hours. In the absence of hypotension and other complications, by the second or third day patients typically are ambulating in their room with increasing duration and frequency. By day 3 after infarction, patients should be increasing their ambulation progressively to a goal of 185m atleast three times a day.

Diet:

Because of the risk of emesis and aspiration soon after STEMI, patients should receive either nothing or only clear liquids by mouth for the first 4 – 12 hours. Portions should not be large and the menu should be enriched with foods that are high in potassium, magnesium and fibre but low in sodium.

Bowels:

Bed rest and the effect of narcotics used for the relief of pain often lead to constipation. A bedside commode rather than a bedpan, a diet rich in bulk and the routine use of a stool softener are recommended.

Sedation:

Many patients require sedation during hospitalisation to withstand the period of enforced inactivity with tranquility.

Pharmacotherapy:

Antithrombotic agents:

The use of antiplatelet and antithrombotic therapy during the initial phase of STEMI is based on extensive laboratory and clinical

evidence that thrombosis plays an important role in the pathogenesis of this condition.

1. The primary goal of treatment with antiplatelet and antithrombotic agents is to establish and maintain patency of the infarct related artery.

2. The secondary goal is to reduce the patients tendency to thrombosis and thus the likelihood of mural thrombus

The most compelling evidence for the benefits of antiplatelet therapy mainly with aspirin in STEMI is found in the comprehensive overview by the Antiplatelet Trialist Collaboration. Data from nearly 20,000 patients with MI enrolled in 15 randomized trials were pooled and revealed a relative reduction of 27% in the mortality rate, from 14.2% in control patients to 10.4% in patients receiving antiplatelet therapy. The addition of clopidogrel to background treatment with aspirin to STEMI patients reduces the risk of death, reinfarction or stroke.

The standard antithrombotic agent used in clinical practice is unfractionated heparin. The recommended dose of unfractionated heparin is an initial bolus of 60U/Kg followed by an initial infusion of 12U/Kg/hour. The activated partial thromboplastin time during maintenance therapy should be 1.5- 2 times the control value.

Patients with anterior location of the infarction, severe LV dysfunction, heart failure, a history of embolism, two dimensional echocardiographic evidence of mural thrombus or atrial fibrillation are at increased risk of systemic or pulmonary thromboembolism. Such individuals should receive full therapeutic levels of antithrombotic therapy followed by atleast three months of warfarin therapy.

β adrenoreceptor blockers:

Beta blocker therapy after STEMI is useful for most patients except those in whom it is specifically contraindicated such as patients with heart failure or severly compromised LV function, heart block, orthostatic hypotension or a history of asthma and perhaps those whose excellent prognosis defined as an expected mortality rate of <1% peryear, patients<55 years, no previous MI, with normal ventricular function, no complex ventricular ectopy and no angina markedly diminishes any potential benefit.

Inhibition of Renin Angiotensin- Aldosterone system:

ACE inhibitors reduce the mortality rate after STEMI and the mortality benefits are additive to those achieved with aspirin and beta blockers. Long term aldosterone blockade should be prescribed for STEMI patients without significant renal dysfunction (creatinine

≥2.5mg/dl in men and ≥2.0mg/dl in women) or hyperkalemia (potassium ≥5.0meq/L) who are already receiving therapeutic doses of an ACE inhibitors.

Complications:

1. Ventricular dysfunction:

The overall chamber enlargement that occurs is related to the size and location of the infarct, with greater dilatation following infarction of the anterior wall and apex of the left ventricle and causing more marked hemodynamic impairment, more frequent heart failure and a poorer prognosis. A classification originally proposed by Killip divides patients into four groups

Class I: No signs of pulmonary or venous congestion.

Class II: Moderate heart failure as evidenced by rales at the lung bases, S_3 gallop, tachypnea or signs of right heart failure including venous and hepatic congestion.

Class III: Severe heart failure, pulmonary edema.

Class IV: shock with systolic pressure <90mmHg and evidence of peripheral vasoconstriction, peripheral cyanosis, mental confusion and oliguria.

2. Cardiogenic shock

3. Arrhythmias:

The prompt management of arrhythmias constitutes a significant advance in the treatment of STEMI. Pharmacological therapy is now reserved for patients with sustained ventricular arrhythmias

1. Ventricular tachycardia and fibrillation
2. Accelerated idioventricular rhythm
3. Supraventricular arrhythmias
4. Atrioventricular and interventricular conduction disturbances

4. Pericarditis

5. Thromboembolism

6. Left ventricular aneurysm

Post infarction risk stratification and management:

Many clinical and laboratory factors have been identified that are associated with an increase in cardiovascular risk after initial recovery from STEMI. Some of the most important risk factors include

1. Persistent ischemia (spontaneous or provoked)
2. Depressed LV ejection fraction <40%
3. Rales above the lung base on physical examination or congestion on chest radiograph
4. Symptomatic ventricular arrhythmias
5. Age >75 years
6. Diabetes
7. History of previous myocardial infarction
8. Hypotension

The usual duration of hospitalization for an uncomplicated STEMI is about 5 days. During the first 1- 2 weeks, the patient should be encouraged to increase activity by walking. After 2 weeks, the physician must regulate the patient's activity on the basis of exercise tolerance. Most patients will be able to work within 2-4 weeks.

MATERIALS AND METHODS

Hundred women admitted with acute myocardial infarction in coronary care unit of Govt General Hospital were randomly selected for the study for a period of one year from July 2008 to June 2009.

Persons included in the study were informed about the aim of the study and consent was obtained.

Information like age, symptoms, time interval to reach hospital, associated comorbid conditions such as hypertension, diabetes, coronary artery disease, family history, menstrual status, complete clinical examination and treatment details were collected.

Investigations like electrocardiogram, echocardiogram, renal function test, lipid profile were also done.

All the patients were followed up during their hospital stay and the outcome recorded.

The collected data were analysed with regards to age of presentation, menstrual status, symptoms, time to reach hospital since the onset of symptoms, severity of clinical presentation according to Killip's

classification, electrocardiographic changes and echocardiographic changes with in hospital outcome.

Pump failure is now the primary cause of in hospital death due to STEMI. The extent of infarction correlates well with the degree of pump failure and with mortality both early and later[2].

OBSERVATIONS

Among the hundred women selected for the study, 21 were in the age group of 50 and below, 33 were in the 51 to 60 age group, 22 were in the 61 to 70 age group and 24 were in the age group of above 70. This is shown in Table 1 and Figure 1. The mean age is 60.97 and the range is 35 – 85.

Age wise distribution of cases:

Age in years	**Frequency (n = 100)**
50 and below	21
51 – 60	33
61 – 70	22
Above 70	24

Table- 1

The interheart study which was a case control study with 15152 cases of acute MI and 14820 controls from 262 centres of 52 countries

addressed the specific question of why have their first MI 9 to 10 years after men and concluded that younger women have lower risk factors[6].

Of the total, 43 women reached the hospital with a time interval of 6 hours or less since the onset of symptoms. 57 of the total took more than 6 hours to reach the hospital. This is shown in Table 2 and Figure 2.

Time interval to reach hospital:

Time interval to reach hospital	Frequency (n = 100)
6 hrs or less	43
More than 6 hrs	57

Table - 2

VVS Bonerjee et al state that time from onset of symptoms to randomization was significantly longer in women indicating a modest delay in time to treatment in females[7].

Of the total, 43 women experienced typical symptoms suggestive of angina. Remaining 57 presented with atypical symptoms. This is shown in Table 3.

Symptoms:

Symptoms	Frequency (n=100)
Typical	43
Atypical	57

Table – 3

Sheps DS et al conclude that the typical textbook symptoms of substernal chest pain or pressure radiating to the arms may not be the primary symptoms in women presenting with coronary artery disease. Other symptoms such as dyspnea, fatigue and lack of energy may be predominant[3]. The new onset of symptoms and their relationship to activity has more clinical relevance than their physical location[8].

Moreover the Rotterdam study found that the proportion of unrecognized MI in women was more than 54%[9]. The Framingham study also reported a higher proportion of unrecognized MI in women[10].

In this study 32 women had no risk factors at all. 49 had atleast one risk factor and 19 had two risk factors that can predispose to coronary artery disease. This is shown in Table 4.

Risk factors:

Risk factors	Frequency (n=100)
0 risk factor	32
1 risk factor	49
2 risk factors	19

Table – 4

Yusuf et al, state that, for women hypertension and diabetes may need to be treated more aggressively. There should be greater attention to increased physical activity as a strategy to prevent diabetes[6].

Schatzkin et al conclude that in women, traditional risk factors have distinct effect on the mechanism of sudden coronary death. Effective risk factor modification may therefore differ between younger and older women and may be targeting different mechanisms of plaque instability[11].

Out of the total, 26 had family history of risk factors. Remaining 74 did not have any family history of risk factors. This is shown in Table 5.

Family history of risk factors:

Family history of risk factors	Frequency (n=100)
Absent	74
Present	26

Table - 5

In the study group 88 attained menopause and 12 were menstruating. This is shown in Table 6.

Menstrual status:

Menstrual status	Frequency (n=100)
Menstruating	12
Menopause	88

Table – 6

Rexrode et al state that among women, low SHBG levels were a marker of cardiovascular risk but this risk was not independent of BMI, hypertension and diabetes[12].

Patients were grouped using Killip classification of pump failure. 45 women were grouped under class I, 26 women under class II, 28 under class III and one under class IV. This is shown in Table 7.

Distribution of cases according to Killip classification:

Killip class	Frequency (n=100)
I	45
II	26
III	28
IV	1

Table – 7

Irrespective of the other risk factors, 88 women had a normal lipid profile and 12 women had an abnormal lipid profile. This is shown in Table 8.

Lipid abnormalities:

Lipid profile	Frequency (n=100)
Normal	88
Abnormal	12

Table – 8

In the GUSTO II ACS study, involving more than 12,000 patients, women were older at presentation for ACS and had a higher prevalence of risk factors such as hypertension, diabetes and hypercholestrolemia[13].

According to the electrocardiographic manifestations, 11 women suffered anterolateral MI, 41 suffered anterior wall MI, 2 suffered high lateral MI, 29 had inferior wall MI, 3 had inferior and posterior wall MI, 8 had inferior wall and right ventricular MI, 6 women had inferior wall, right ventricular and posterior wall MI. This is shown in Table 9 and Figure 3.

Electrocardiographic manifestations:

Type of MI by ECG	Frequency (n=100)
ASMI	11
AWMI	41
HLMI	2
IWMI	29
IW&PWMI	3
IW&RVMI	8
IW&RV&PWMI	6

Table – 9

Echocardiographic assessment of left ventricular function revealed that 20 women had severe LV dysfunction, another 20 had moderate LV dysfunction, 22 had mild LV dysfunction, and 21 had regional wall

motion abnormality. Out of the studied people 14 women did not have any regional wall motion abnormality. This is shown in Table 10.

Echocardiographic manifestations:

LV function by ECHO	Frequency (n=100)
No RWMA	14
RWMA	21
Diastolic dysfunction	3
Mild LV dysfunction	22
Moderate LV dysfunction	20
Severe LV dysfunction	20

Table - 10

55 women had the eligibility of being treated with thrombolytic therapy. 45 women were treated with heparin. This is shown in Table 11.

Treatment given:

Treatment given	Frequency (n=100)
SK	55
Heparin	45

Table – 11

On short term follow up 78 recovered and 22 succumbed to death. This is shown in table 12.

Outcome:

Outcome	Frequency (n=100)
Recovered	78
Death	22

Table – 12

On comparing age of presentation to the outcome on short term follow up, 15 deaths occurred in those above 60 years of age and 7 deaths occurred among aged 60 and below. This is statistically significant. This is shown in table 13 and Figure 4.

Comparing age and outcome:

Age in years	**Frequency (n=100)**	**Outcome**		**p value**	**Significance**
		Recovered	**Death**		
≤ 60 yrs	54	47	7	0.0281	Significant
> 60 yrs	46	31	15		

Table - 13

Hochman et al state that crude rates of death at 30 days were significantly higher for women[14]. Maynard et al have also reported that women with acute myocardial infarction have high in hospital mortality. Women were older and had more co existing conditions. After adjustment for such differences, many studies have concluded that sex is not an independent predictor of mortality after acute myocardial infarction[15].

GUSTO II b data demonstrate that although women with acute coronary syndromes have increased mortality. The reason for increased mortality appears age dependent rather than gender dependent[16].

When the duration to reach hospital after the onset of symptoms is compared with outcome, 18 deaths occurred in the group of women who reached hospital more than 6 hours from the onset of symptoms whereas only 4 deaths occurred in those who reached the hospital within 6 hours of onset of symptoms. This is statistically significant. This is shown in table 14 and Figure 5.

Comparing duration to reach hospital and outcome:

Duration to reach hospital	**Frequency (n=100)**	**Outcome**		**p value**	**Significance**
		Recovered	**Death**		
≤ 6 HRS	43	39	4	0.0080	Significant
> 6 HRS	57	39	18		

Table – 14

Mosca et al suggest that the traditional role of the female as the care giver rather than care seeker has been implicated in their reluctance to seek medical assistance for the atypical symptoms that they often develop. Also NSTEMI's are more common in women. This clearly adds to the difficulty in diagnosing AMI in women and further confounds delay to initiation of appropriate treatment[8, 17].

When the mortality is compared with the presence of risk factors higher mortality i.e. 26.3% was noted when 2 risk factors are present. Whereas it is 15.63% when there is no risk factor and 24.5% in the presence of one risk factor. This is shown in Table 15.

Risk factors and outcome:

Risk factors	**Frequency (n=100)**	**Outcome**	
		Recovered	**Death**
0 risk factor	32	27	5 (15.63%)
1 risk factor	49	37	12 (24.5%)
2 risk factors	19	14	5 (26.3%)

Table - 15

Anand et al state that higher mortality in women after acute myocardial infarction is significantly related to the presence of multiple coexisting conditions like diabetes, hypertension, measured waist / hip ratio, physical activity etc[18,19].

29.82% death was recorded in those who presented with atypical symptoms and only 11.62% died of those who presented with typical symptoms. This is statistically significant. This is shown in table 16 and Figure 6.

Symptoms and outcome:

Symptoms	**Frequency (n=100)**	**Outcome**		**p value**	**Significance**
		Recovered	**Death**		
Typical	43	38	5	0.0495	Significant
Atypical	57	40	17		

Table – 16

22.97% deaths occurred in those women who did not have a family history of risk factors. 19.2% deaths occurred in those women who had a family history of risk factors. This is shown in Table 17.

Family history and outcome:

Family history of risk factors	**Frequency (n=100)**	**Outcome**	
		Recovered	**Death**
Absent	74	57	17(22.97%)
Present	26	21	5 (19.2%)

Table – 17

When the outcome is compared with killip classification 100% deaths occurred in those with class IV, 71.4% deaths occurred in class III, 0% in class II and 2.2% deaths in those with class I was observed. This is shown in Table 18.

Killip class and outcome:

Killip class	Frequency (n=100)	Outcome	
		Recovered	Death
I	45	44	1 (2.2%)
II	26	26	0 (0%)
III	28	8	20(71.4%)
IV	1	0	1(100%)

Table –18

On comparing outcome with type of myocardial infarction, 9.1% deaths ASMI, 19.5% deaths in AWMI, 10.3% in IWMI, 33.33% in IW&PWMI, 75% deaths in IW&RVMI, 50% in IW, RV&PWMI were observed. This is shown in Table 19.

Type of MI and outcome:

Type of MI by ECG	Frequency (n=100)	Outcome	
		Recovered	Death
ASMI	11	10	1 (9.1%)
AWMI	41	33	8 (19.5%)
HLMI	2	2	0 (0%)
IWMI	29	26	3 (10.3%)
IW&PWMI	3	2	1(33.33%)
IW&RVMI	8	2	6 (75%)
IW&RV&PWMI	6	3	3 (50%)

Table - 19

On studying left ventricular dysfunction, 85% deaths occurred in those with severe LV dysfunction. 33.3% in those with diastolic

dysfunction, 10% in those with moderate LV dysfunction, 9.5 % in those with regional wall motion abnormalities were observed. This is shown in Table 20.

LV function and outcome:

LV function by ECHO	**Frequency (n=100)**	**Outcome**	
		Recovered	**Death**
No RWMA	14	14	0 (0%)
RWMA	21	19	2 (9.5%)
Diastolic dysfunction	3	2	1(33.33%)
Mild LV dysfunction	22	22	0 (0%)
Moderate LV dysfunction	20	18	2 (10%)
Severe LV dysfunction	20	3	17 (85%)

Table – 20

DISCUSSION

Cardiovascular disease is the leading cause of mortality in women. In fact CVD is responsible for a third of all deaths of women worldwide and half of all deaths of women over 50 years of age in developing countries[20, 21]. Retrospective analysis suggest that there are some clinically relevant differences between women and men in terms of prevalence, presentation, management and outcomes of the disease, but little is known about why CVD affects women differently. Historically, women have been under represented in clinical trials. The lack of good trial evidence concerning sex specific outcomes has led to the assumption about CVD treatment in women, which in turn may have resulted in inadequate diagnosis and sub optimal management, greatly affecting outcomes. This knowledge gap may also explain why cardiovascular health in women is not improving as fast as that of men[22, 23]. Over the last decades, mortality rates in men have steadily declined, while those in women remained stable. It is also becoming increasingly evident that gender differences in cultural, behavioral, psychosocial and socio economic status are responsible to various degrees, for the observed differences in women. However the interaction between sex and gender related factors and CVD outcomes in women remains largely unknown[24, 25,26].

This study is a cross sectional observational study. So it has its own limitations. Cross sectional studies are susceptible to several sources of bias and confounding. The study concentrates about the age of presentation, symptoms, delay in treatment, clinical profile, complications and short term mortality.

Hundred patients admitted to coronary care unit with evidence of acute myocardial infarction features in electrocardiogram were randomly selected and data collected with the follow up during the hospital stay of patients.

In this study the mean age is 60.97 and the range is 35 – 85. Total mortality is 22% and there is a significant correlation between mortality and age. Women of advanced age develop more complications and mortality. Johanne Neil et al state that the excess mortality in women is due to older age at presentation in women[27, 28].

63% women presented with atypical symptoms. This study concludes that women classically present at an advanced age, with atypical symptoms resulting in a delay of initiation of treatment or inadequate treatment leading to poor short term outcome. There is a significant delay in reaching the treatment care center. In this study there is significant correlation between time to receive treatment since the onset of symptoms and poor short term outcome. Sandra et al state that, women

receive somewhat less aggressive treatment during the early management of acute myocardial infarction[29, 30]. Hani Jneid et al state that the under use of evidence based treatments and delayed reperfusion among women represent potential opportunities for reducing poor outcomes after AMI[31].

Those women who developed MI at an age younger than 60 years had more than one risk factor. Risk factors such as diabetes, hypertension play an important role in predisposing younger women to CAD. So aggressive treatment of diabetes and hypertension should be encouraged in women. This is consistent with previous studies. Burke et al state that, in women traditional risk factors have distinct effects on the mechanism of sudden coronary death, which vary by menopausal status. Effective risk factor modification may therefore differ between younger and older women and may targeting different mechanisms of plaque rupture[32, 33].

88% of women were postmenopausal. Significant percentage of women presented with advanced stages in Killip classification.

The most common pattern of myocardial infarction is anterior wall myocardial infarction.62% of women had Lvdysfunction and out of them 20% had severe LV dysfunction. 55% were eligible enough to receive adequate thrombolytic therapy.

CONCLUSION

1. Women with advanced age have a poorer outcome.

2. Women clinically present with atypical symptoms that has resulted in a significant delay to reach treatment centre.

3. In hospital mortality is directly related to atypical symptoms, delay in reaching the hospital and co morbidities.

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PROFORMA

SERIAL NO:

NAME: AGE: SEX:

ADDRESS:

OCCUPATION: INCOME:

IP NO: DOA: DOD:

TIME INTERVAL TO REACH HOSPITAL:

COMPLAINTS:

PRESENT HISTORY: Chest Pain - Typical/Atypical
Palpitation
Syncope
Dyspnea
PND, Orthopnea
Giddiness
Nausea, Vomiting

PAST HITORY: DM/HT/IHD/CVA

FAMILY HISTORY: DM/HT/CAD/Premature IHD

PERSONAL HISTORY: Smoking/Alcoholic/Diet/Oral Contraceptive Pills/HRT

MENSTRUAL HISTORY:

EXAMINATION:

General Examination:

Anemia/Jaundice/Pedal edema/ Dyspnea

Pulse: BP: JVP:

Cardiovascular System Examination:

Heart sounds:

Murmur:

Pericardial rub:

Killip class:

Respiratory System Examination:

Abdomen Examination:

Central Nervous System Examination:

INVESTIGATIONS:

Urine - Albumin
Sugar
Deposits

Blood - Sugar
Urea
Creatinine

Lipid Profile- Total Cholesterol
TGL
LDL
HDL
VLDL

Chest X Ray:

ECG:

ECHO:

TREATMENT GIVEN:

OUTCOME:

ABBREVIATIONS

CVD	-	Cardio vascular disease
CAD	-	Coronary artery disease
IHD	-	Ischemic heart disease
ECG	-	Electrocardiogram
STEMI	-	ST elevation MI
MI	-	Myocardial infarction
DASH	-	Dietary approaches to stop hypertension
BP	-	Blood pressure
HDL	-	High density lipoprotein
LDL	-	Low density lipoprotein
UA	-	Unstable angina
CK	-	Creatine kinase
CK – MB	-	Creatine kinase - MB
Tc	-	Technitium
ACC	-	American college of cardiology
AHA	-	American heart association
EMS	-	Emergency medical services
PCI	-	Percutaneous coronary intervention
CPR	-	Cardio pulmonary resuscitation

INR	-	International normalized ratio
TNK	-	Tenecteplase
rPA	-	Reteplase
ACE	-	Angiotensin converting enzyme
LV	-	Left ventricle
SHBG	-	Sex hormone binding globulin
BMI	-	Body mass index
RWMA	-	Regional wall motion abnormality
SK	-	Streptokinase
ACS	-	Acute coronary syndrome
AWMI	-	Anterior wall myocardial infarction
ASMI	-	Antero septal myocardial infarction
IWMI	-	Inferior wall myocardial infarction
RVMI	-	Right ventricular myocardial infarction
PWMI	-	Posterior wall myocardial infarction
HLMI	-	High lateral myocardial infarction

Figure – 1

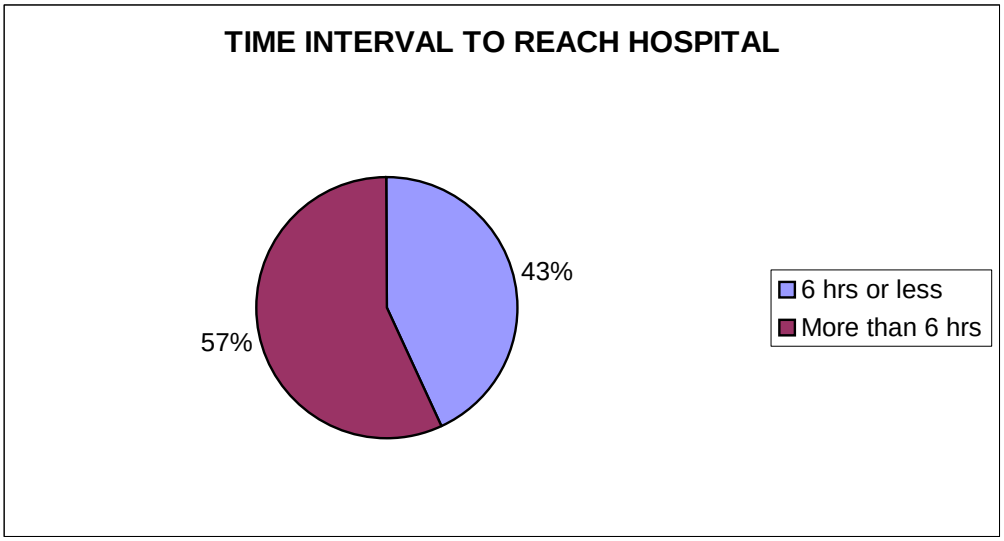

Figure -2

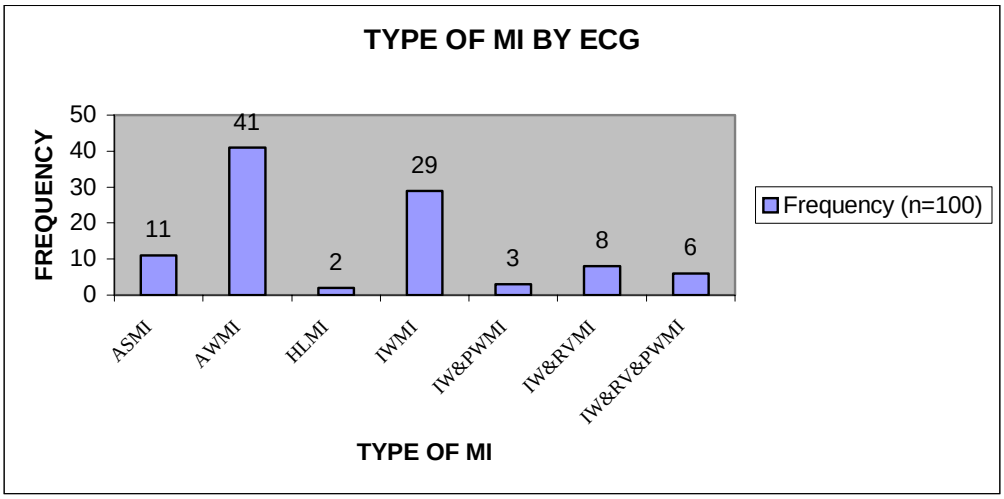

Figure - 3

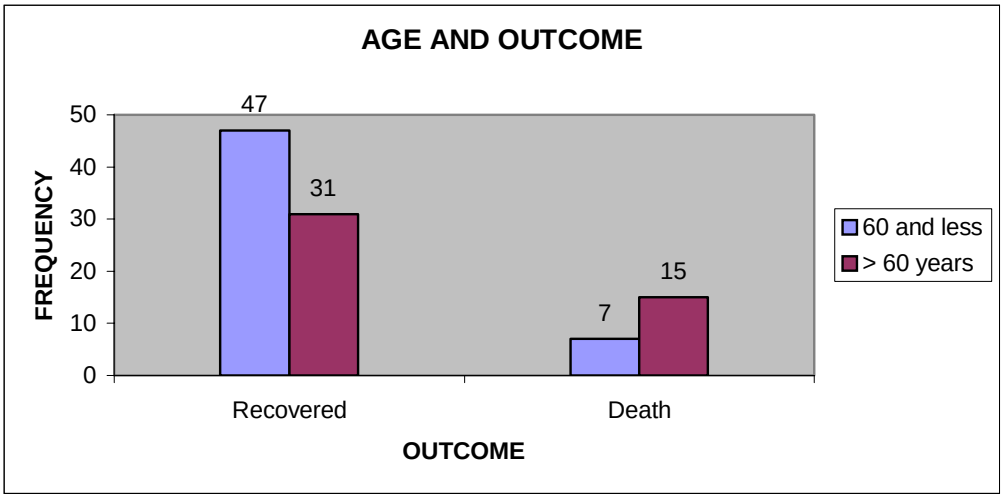

Figure - 4

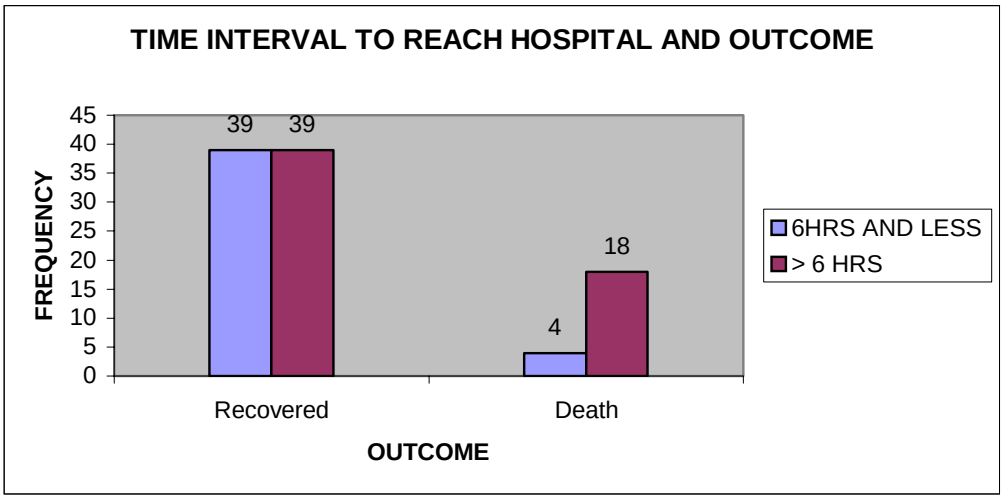

Figure - 5

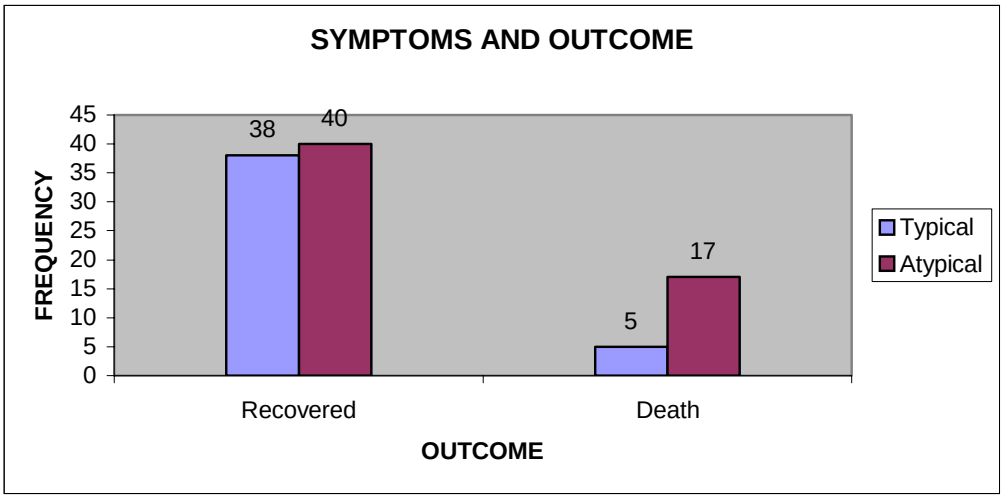

Figure - 6

MASTER CHART

Serial No	Age	Time interval to reach hospital	Symptoms Typical/ Atypical	Past history DM/HT/ CAD/CVA	Family history DM/HT/ CAD	Menstrual history	Killip class	Lipid profile	ECG	ECHO	Treatment given	Outcome
1	46	01 HR	Typical	DM	DM	MP	I	TGL	IWMI&RVMI	RWMA	SK	RECOVERED
2	70	08 HRS	Atypical	HT	NIL	MP	I	N	IWMI	NO RWMA	SK	RECOVERED
3	69	12 HRS	Atypical	DM/HT	DM,CAD	MP	III	TGL	IWMI	DD	HEPARIN&NTG	DEATH
4	50	72 HRS	Atypical	DM	NIL	MP	III	N	AWMI	RWMA	HEPARIN&NTG	RECOVERED
5	49	96 HRS	Atypical	DM/HT	DM,HT	MP	II	TGL,LDL	AWMI	MILD LV DYSFN	HEPARIN	RECOVERED
6	48	48 HRS	Typical	NIL	NIL	MP	I	N	ASMI	MOD LV DYS	HEPARIN	RECOVERED
7	67	96 HRS	Atypical	DM	NIL	MP	III	N	AWMI	RWMA&VSR	HEPARIN	DEATH
8	35	04 HRS	Atypical	DM/IHD	DM	M	II	TGL	HLMI	RWMA	SK	RECOVERED
9	75	04 HRS	Atypical	DM/HT	DM	MP	III	N	AWMI	SEV LV DYSFN	SK	DEATH
10	75	12 HRS	Atypical	HT	NIL	MP	III	N	IWMI	MILD LV DYSFN	HEPARIN	RECOVERED
11	75	12 HRS	Atypical	CAD	NIL	MP	IV	N	IWMI&RVMI	SEV LV DYSFN	SK	DEATH
12	45	04 HRS	Typical	HT	HT	M	I	TGL	AWMI	MILD LV DYSFN	SK	RECOVERED
13	57	02 HRS	Typical	NIL	NIL	MP	I	N	AWMI	RWMA	SK	RECOVERED
14	70	08 HRS	Typical	NIL	NIL	MP	I	N	AWMI	RWMA	SK	RECOVERED
15	56	14 HRS	Atypical	DM/HT	NIL	MP	I	N	IWMI	NO RWMA	HEPARIN	RECOVERED
16	75	10 HRS	Typical	NIL	NIL	MP	II	N	AWMI	MILD LV DYSFN	HEPARIN	RECOVERED
17	60	06 HRS	Atypical	DM/HT	DM	MP	III	N	IWMI	NO RWMA	SK	RECOVERED
18	47	04 HRS	Atypical	DM/HT	NIL	M	I	TGL,LDL	ASMI	NO RWMA	SK	RECOVERED
19	55	12 HRS	Atypical	NIL	CAD	MP	II	N	AWMI	MILD LV DYSFN	HEPARIN&NTG	RECOVERED
20	57	14 HRS	Atypical	HT	NIL	MP	I	N	IWMI	DD	HEPARIN	RECOVERED
21	70	04 HRS	Atypical	DM/HT	NIL	MP	III	N	IW&PW&RVMI	MOD LV DYS	SK	DEATH
22	57	12 HRS	Atypical	NIL	CAD	MP	I	TGL	ASMI	MILD LV DYSFN	HEPARIN	RECOVERED
23	45	06 HRS	Atypical	DM/HT	NIL	M	I	TGL	IWMI	RWMA	SK	RECOVERED
24	47	24 HRS	Atypical	DM/HT	NIL	M	III	N	IWMI	DD	HEPARIN	RECOVERED
25	60	48 HRS	Atypical	HT	NIL	MP	III	N	IW&PW&RVMI	SEV LV DYSFN	HEPARIN	DEATH
26	60	20 HRS	Atypical	DM	NIL	MP	III	N	IW&PW&RVMI	MILD LV DYSFN	HEPARIN	RECOVERED
27	68	48 HRS	Atypical	DM	NIL	MP	II	N	IWMI	RWMA	HEPARIN	RECOVERED
28	56	06 HRS	Typical	HT	NIL	MP	II	N	IWMI	MILD LV DYSFN	SK	RECOVERED
29	40	24 HRS	Atypical	DM	DM	M	II	LDL	AWMI	MOD LV DYS	HEPARIN	RECOVERED
30	60	06 HRS	Typical	HT	NIL	MP	III	N	AWMI	SEV LV DYSFN	SK	RECOVERED

31	55	20 HRS	Atypical	HT/CAD	CAD	MP	I	N	ASMI	RWMA	HEPARIN	RECOVERED
32	65	20 HRS	Atypical	HT/CAD	HT	MP	II	N	AWMI	MILD LV DYSFN	HEPARIN	RECOVERED
33	43	22 HRS	Atypical	DM	DM	MP	III	TGL	AWMI	MOD LV DYS	HEPARIN	DEATH
34	73	28 HRS	Typical	NIL	NIL	MP	III	N	IW&RVMI	SEV LV DYSFN	HEPARIN	DEATH
35	45	04 HRS	Typical	HT	CAD	M	I	N	IWMI	NO RWMA	SK	RECOVERED
36	63	48 HRS	Atypical	NIL	NIL	MP	I	N	AWMI	MILD LV DYSFN	HEPARIN	RECOVERED
37	62	06 HRS	Typical	HT	NIL	MP	I	N	ASMI	NO RWMA	HEPARIN	RECOVERED
38	35	02 HRS	Typical	HT	CAD	M	I	N	AWMI	RWMA	SK	RECOVERED
39	45	06 HRS	Typical	CAD	NIL	M	II	TGL,LDL	IWMI	MILD LV DYSFN	SK	RECOVERED
40	50	12 HRS	Atypical	DM	DM	M	III	TGL	IWMI&PWMI	SEV LV DYSFN	HEPARIN	DEATH
41	76	06 HRS	Typical	NIL	NIL	MP	II	N	AWMI	MOD LV DYS	SK	RECOVERED
42	60	02 HRS	Typical	HT	NIL	MP	I	N	AWMI&LBBB	MILD LV DYSFN	SK	RECOVERED
43	57	02 HRS	Typical	DM	NIL	MP	II	N	IWMI	RWMA	SK	RECOVERED
44	80	12 HRS	Atypical	NIL	NIL	MP	III	N	AWMI	SEV LV DYSFN	HEPARIN	DEATH
45	54	10 HRS	Atypical	DM	NIL	MP	I	N	AWMI	NO RWMA	HEPARIN	RECOVERED
46	81	20 HRS	Atypical	HT	NIL	MP	I	N	IWMI	NO RWMA	HEPARIN	RECOVERED
47	54	12 HRS	Atypical	HT/DM	NIL	MP	III	N	IW&RV& PWMI	SEV LV DYSFN	HEPARIN	DEATH
48	72	02 HRS	Typical	HT	NIL	MP	III	N	IWMI	SEV LV DYSFN	SK	DEATH
49	85	10 HRS	Atypical	DM	NIL	MP	III	N	IWMI&CHB	SEV LV DYSFN	HEPARIN	DEATH
50	57	06 HRS	Typical	NIL	NIL	MP	II	N	AWMI	MOD LV DYS	SK	RECOVERED
51	45	02 HRS	Typical	NIL	NIL	MP	I	N	IWMI	RWMA	SK	RECOVERED
52	74	04 HRS	Typical	NIL	NIL	MP	I	N	AWMI	MILD LV DYSFN	SK	RECOVERED
53	57	12 HRS	Atypical	HT	NIL	MP	I	N	AWMI	MILD LV DYSFN	HEPARIN	RECOVERED
54	55	10 HRS	Atypical	DM	DM	MP	I	N	AWMI	MILD LV DYSFN	HEPARIN	RECOVERED
55	62	12 HRS	Atypical	DM/HT	NIL	MP	III	N	AWMI	SEV LV DYSFN	HEPARIN	DEATH
56	76	10 HRS	Atypical	DM	NIL	MP	III	N	AWMI	SEV LV DYSFN	HEPARIN	DEATH
57	55	24 HRS	Atypical	DM/HT	HT	MP	III	N	AWMI&CHB	SEV LV DYSFN	HEPARIN	RECOVERED
58	39	02 HRS	Typical	DM/HT	DM,HT	M	II	N	AWMI	MOD LV DYS	SK	RECOVERED
59	52	06 HRS	Typical	HT	HT	MP	I	N	IWMI	RWMA	SK	RECOVERED
60	56	07 HRS	Atypical	HT	NIL	MP	I	N	AWMI	RWMA	SK	DEATH
61	75	08 HRS	Typical	NIL	NIL	MP	III	N	AWMI	SEV LV DYSFN	SK	DEATH
62	72	08 HRS	Typical	NIL	NIL	MP	III	N	AWMI	SEV LV DYSFN	SK	RECOVERED
63	60	02 HRS	Typical	HT	NIL	MP	II	N	ASMI	MOD LV DYS	SK	RECOVERED
64	53	02 HRS	Atypical	DM	DM,CAD	MP	I	N	IW&PWMI	MOD LV DYS	SK	RECOVERED
65	65	06 HRS	Typical	NIL	NIL	MP	II	N	IW&PW&RVMI	MILD LV DYSFN	SK	RECOVERED
66	75	12 HRS	Atypical	NIL	NIL	MP	II	N	AWMI	MOD LV DYS	SK	RECOVERED

67	65	02 HRS	Typical	NIL	NIL	MP	I	N	AWMI	MILD LV DYSFN	SK	RECOVERED
68	76	12 HRS	Atypical	DM/HT	NIL	MP	I	N	AWMI	RWMA	HEPARIN	RECOVERED
69	65	10 HRS	Atypical	NIL	NIL	MP	I	N	AWMI	RWMA	SK	RECOVERED
70	45	12 HRS	Atypical	DM	NIL	MP	I	N	ASMI	NO RWMA	HEPARIN	RECOVERED
71	63	02 HRS	Typical	HT	NIL	MP	I	N	ASMI	NO RWMA	SK	RECOVERED
72	65	10 HRS	Atypical	NIL	CAD	MP	I	N	IW&PWMI	RWMA	SK	RECOVERED
73	49	02 HRS	Typical	CAD	NIL	MP	II	N	IW&PW&RVMI	MOD LV DYS	SK	RECOVERED
74	43	12 HRS	Typical	HT	NIL	M	I	N	IWMI	NO RWMA	HEPARIN	RECOVERED
75	55	07 HRS	Atypical	HT	NIL	MP	III	N	IW&RVMI&AF	SEV LV DYSFN	SK	DEATH
76	52	07 HRS	Typical	HT	NIL	MP	II	N	IW&RWMI	MOD LV DYS	SK	RECOVERED
77	75	10 HRS	Atypical	NIL	NIL	MP	II	N	IWMI	MOD LV DYS	SK	RECOVERED
78	61	06 HRS	Atypical	HT	NIL	MP	I	N	ASMI	MILD LV DYSFN	SK	RECOVERED
79	60	02 HRS	Typical	NIL	DM	MP	I	N	IWMI	NO RWMA	SK	RECOVERED
80	60	04 HRS	Typical	DM	DM	MP	III	N	ASMI	SEV LV DYSFN	SK	DEATH
81	57	02 HRS	Atypical	DM/HT	NIL	MP	I	N	AWMI	RWMA	SK	RECOVERED
82	85	02 HRS	Atypical	NIL	NIL	MP	I	N	IWMI	NO RWMA	SK	RECOVERED
83	65	12HRS	Atypical	NIL	NIL	MP	II	N	AWMI	MOD LV DYS	HEPARIN	RECOVERED
84	75	02 HRS	Typical	NIL	NIL	MP	II	N	AWMI	MOD LV DYS	SK	RECOVERED
85	48	02 HRS	Typical	HT	NIL	MP	I	N	ASMI	RWMA	SK	RECOVERED
86	52	12 HRS	Atypical	HT	NIL	MP	I	N	IWMI	NO RWMA	HEPARIN	RECOVERED
87	70	12 HRS	Typical	NIL	NIL	MP	II	N	IWMI	MOD LV DYS	HEPARIN	RECOVERED
88	70	20 HRS	Atypical	NIL	NIL	MP	III	N	IW&RVMI	SEV LV DYSFN	HEPARIN	DEATH
89	80	24 HRS	Atypical	HT	NIL	MP	III	N	IW&RVMI&CHB	SEV LV DYSFN	SK	DEATH
90	75	02 HRS	Atypical	HT	NIL	MP	I	N	IWMI	MILD LV DYSFN	SK	RECOVERED
91	65	12 HRS	Typical	NIL	NIL	MP	III	N	IW&RVMI	SEV LV DYSFN	HEPARIN	DEATH
92	70	12 HRS	Atypical	NIL	NIL	MP	II	N	IWMI	MOD LV DYS	HEPARIN	RECOVERED
93	72	10 HRS	Atypical	NIL	NIL	MP	II	N	AWMI	MOD LV DYS	HEPARIN	RECOVERED
94	60	12 HRS	Typical	HT	NIL	MP	I	N	HLMI	MILD LV DYSFN	HEPARIN	RECOVERED
95	70	10 HRS	Atypical	DM	NIL	MP	II	N	IWMI	MOD LV DYS	HEPARIN	RECOVERED
96	75	05 HRS	Typical	NIL	NIL	MP	I	N	AWMI	MILD LV DYSFN	SK	RECOVERED
97	55	06 HRS	Typical	DM/HT	DM,HT	MP	II	N	IWMI	MOD LV DYS	SK	RECOVERED
98	60	02 HRS	Typical	DM	DM	MP	I	N	AWMI	MILD LV DYSFN	SK	RECOVERED
99	72	04 HRS	Typical	NIL	NIL	MP	I	N	IWMI	RWMA	SK	RECOVERED
100	60	02 HRS	Typical	HT	NIL	MP	I	N	AWMI	RWMA	SK	RECOVERED